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## Original Articles.

# PROGRESSIVE SENILE PARAPLEGIA: A CONTRIBUTION TO THE STUDY OF NON-INFLAMMATORY SOFTENING OF THE CORD.[1]

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THE literature of the senile cord is quite meagre. Cruveilhier, Geist, Rokitansky, Durand-Fardel, Empes, are the principal older writers upon the subject. Leyden, in his text-book, has given the most complete description of spinal senile changes.

He shows that there is a tendency for the cord to atrophy slightly and become harder. He finds amyloid bodies very frequently around the central canal and periphery; the cells in the gray matter are somewhat wasted and pigmented, the vessel-walls are thickened and show hyaline degeneration. There seems to be a tendency to stasis of the blood, and foci of arterial thrombosis are found.

The symptoms associated with these changes are: senile tremor, progressive senile weakness and paraplegia, and acute or subacute paralyses from focal softening.

Progressive senile paraplegia in its fully developed form seems to be quite rare. Empes, in 1862 ("Arch. gén. de

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[1] Read at the Annual Meeting of the American Neurological Association, June, 1890.

Médecine"), described what he calls a "progressive senile enfeeblement," which amounted eventually to paraplegia. The exact pathology of this condition has not been firmly established.

The case which I have to report is important, in that it gives evidence of what the pathological condition in these cases is.

It throws some light also on the subject of non-inflammatory softening of the cord.

Systematic writers have generally confessed themselves in the dark as regards the existence of non-inflammatory softening of the cord; and in Gowers' work no mention is made of this subject.

Acute softening has been described usually as synonymous with acute myelitis; but the term is wrongly used, and should not be applied to inflammatory processes at all. Of late it has been suggested that some of the cases of acute myelitis are in fact primarily necrotic processes; but not much evidence has accumulated upon the point.

At all events, the process which I am to describe does not belong to the acute class, but is one strictly of a chronic myelomalacia, and one of a systemic character.

The patient was a man, married, born in Bohemia, seventy years of age; very little known of his early life, except that he was married and had healthy children. About four years ago, at the age of sixty-five or sixty-six, he noticed some weakness in the legs, but he had no pain. This weakness progressed until a year ago, when he was no longer able to walk. About this time he began to have some incontinence of the urine and trouble with the rectal sphincters.

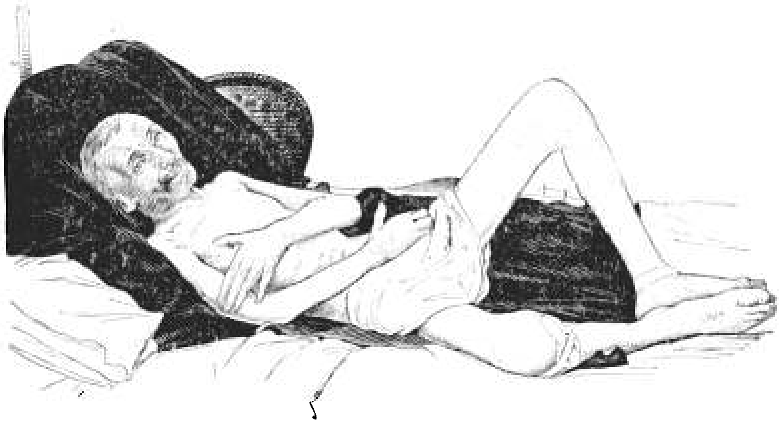
His disease progressed until he entered the Montefiori Home in 1889.

*Status Præsens.*—The patient presents the appearance of senility. He lies in bed with his legs drawn up and nearly immovable. His head is drawn a little to the left. The movements of his arms, face, eyes, and tongue are normal, nor are there any trophic or sensory disturbances here. The disease, except for the slight contracture of the neck, is limited to the lower extremities. These are much wasted and contractured. The knee-jerks are gone, nor is there any clonus or trepidation.

Sensation is normal everywhere; there being, if anything, hyperæsthesia. No pains in the leg or girdle-pains or bed-sores. The urine (which is albuminous) dribbles from the bladder, and there is incontinence of fæces.

The patient's mind was clear, but showed evidences of senility.

The symptoms changed very little, and the patient died finally of exhaustion.



*Autopsy* was made by Dr. Leo Ettinger, house-physician.

*Brain* not examined.

*The Spinal Cord.*—On opening the spinal canal, no apparent abnormalities were observed in the meninges. The cord itself, externally, showed no notable abnormalities to the naked eye. Sections of the fresh cord showed changes in the anterior horns in its lower portion.

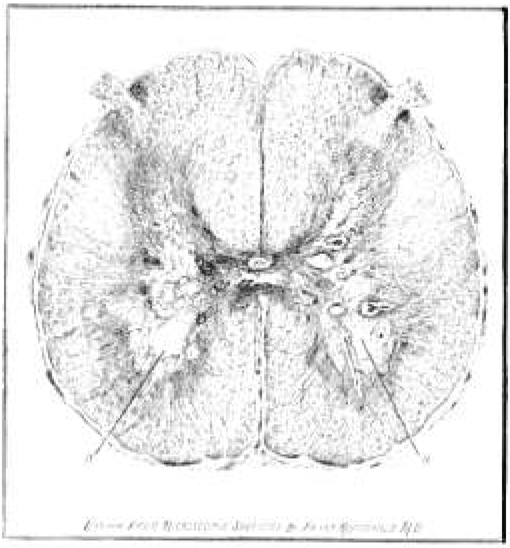
The cord was hardened in Müller's fluid, and stained with hæmatoxylin (Weigert's) and aniline blue.

*Microscopical Examination.*—There was considerable thickening of the pia-mater. Throughout the cord there was seen evidence of marked arterial sclerosis, with thickening of the walls and reduction of cavity, particularly of the smaller vessels.

These changes were most pronounced in the vessels of the anterior median fissure and their branches and in the peripheral arteries radiating toward the anterior horns.

Throughout the cord there was atrophy and absence of the cells of the anterior horns and intermediate gray. In the lumbar region the anterior horns had dropped out, leaving only a ring of healthy gray matter. There was no secondary connective-tissue proliferation and no shrinking of substance.

In the lower dorsal region the anterior horns were reduced to an amorphous granular material thickly interspersed with distended capillaries and here and there a dilated vessel with thickened walls. Ascending the cord, the anterior horns showed more cells, especially upon the periphery, in parts supplied by the peripheral arteries of the pia. In other words, the cerebral arteries were most affected.



The arterial sclerosis affected also the posterior cornua, but to a less extent, and nowhere was there loss of substance.

There was a very noticeable sclerosis in the pyramidal tract on both sides, but more marked high up in the cervical region.

In other respects the white columns were normal. There was loss of fibres in the anterior roots, but not in the posterior.

The anterior cornual cells could be seen in all stages of atrophy, but without any vacuolation or much pigmentation.

The columns of Clark were slightly affected.

The condition, in brief, was one of degenerative endarteritis with sclerosis,obliteration of vessels, causing softening of the anterior horns and intermediate gray matter. This process was accompanied with secondary congestion, dilatation of small vessels and capillaries, but no hemorrhages. The condition was one of softening of the cord precisely analogous to the chronic softening of the brain.

It is not inflammatory, and cannot be called an anterior poliomyelitis. It is not a primary cell-atrophy, and does not, of course, belong to the spinal forms of progressive muscular atrophy.

While the change was, no doubt, largely a senile one, the cord does not correspond to the descriptions of senile cords given by Leyden. Undoubtedly, the feeble gait, loss of knee-jerk, weak bladder, and constipation of senility are dependent upon less marked form of spinal-cord softening than that described here. The disease may be called a progressive senile paraplegia from softening of the anterior horns, due to obliterating arteritis.

Such is my interpretation of the above case; and I have made it while bearing in mind the possible view that the cell-atrophy is primary and the vascular changes secondary.

## INFLUENCE OF THE NERVOUS SYSTEM ON PUTREFACTION.

Ibid. Brown-Sequard sent the following note to the Société de biologie: The head of a guinea-pig was crushed instantly with a hammer and the brain reduced to a jelly. The action of the spinal cord was thus vigorously influenced. Rigor mortis was delayed, and putrefaction did not take place in the intestine till the fourth or fifth day.